

## Early outbreaks of 'epidemic neuromyasthenia'

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### Summary

The literature of the outbreaks of 'epidemic neuromyasthenia' (ENM) from 1934 to 1955 has been selected to show that the disease affects other people besides young adult females in hospitals and nursing homes. There have been district epidemics, in which the male:female ratio was almost even and several male outbreaks affecting soldiers in barracks. Some outbreaks appear to have been triggered off by an epidemic of poliomyelitis, and the epidemiology of outbreaks in Iceland in 1948 and 1955 suggests that the normal cytopathological effects of poliomyelitis infection have been suppressed by the new disease. In the Durban epidemic (1955) a toxic metabolite was discovered in the urine of many patients and a markedly increased urinary excretion of creatine was noted in two New York State outbreaks. The results of the transmission of an agent from patients with ENM to monkeys suggest that the neurological disorder might be in the form of mild disseminated lesions scattered throughout the nervous system from the brain to peripheral nerves and associated with perivascular round cell infiltration without significant cellular damage. ENM infection was widespread in the North of England in 1955 and associated with lymphocyte abnormalities, which have persisted in some cases for several years. This suggests a continuous organic process.

### Introduction

It is over 40 years since descriptions of a new clinical entity began to appear under the headings of 'abortive poliomyelitis' or a 'disease resembling or simulating poliomyelitis'. This illness is now known as 'benign myalgic encephalomyelitis' or 'epidemic neuromyasthenia' (ENM). In the literature of the early epidemics, which occurred up to 1955, there are many observations which not only provide clues to the pathology of the disease but also warrant further investigation by modern research techniques. The first recorded outbreak of ENM occurred in 1934 and provides an introduction to the main clinical features of the disease.

### District and Hospital epidemic in Los Angeles (1934)

The epidemic of poliomyelitis in the Los Angeles area in 1934 was unusual because there was a relatively high attack rate with a low mortality rate (1.4%), a low paralytic rate and a high incidence in adults. A number of outbreaks occurred in institutions with features which differed from those commonly expected in poliomyelitis. The most important epidemic attacked 198 members of the medical and nursing staff of Los Angeles County General Hospital – a case incidence of 4.5%. The main features of the illness in the hospital staff were described by Gilliam (1938) and Marinacci and Von Hagen (1965) as follows:

- (1) the initial systemic and meningeal symptoms were similar to poliomyelitis;
- (2) large diurnal fluctuations of temperature from 97 to 99°F (36.0–37.2°C) were more frequently seen than a rise above 100°F (37.8°C);
- (3) diplopia, constipation and retention of urine were commoner than in poliomyelitis;
- (4) localized muscular weakness occurred in 80% of the cases without the severe degree of atrophy seen in poliomyelitis;
- (5) sensory changes were more pronounced than motor disturbances – especially pain and muscle tenderness, hyperaesthesiae and paraesthesiae, and these symptoms persisted for longer periods than in poliomyelitis;
- (6) mental disturbances, loss of concentration and lapses of memory, sleep disturbances, emotional lability with hysterical episodes and trophic changes were a feature of the disease;
- (7) fatigue on walking short distances and on the least exertion was prominent;
- (8) recurrences of both systemic and neurological symptoms were frequent, and some patients were more disabled by these recurrences than by the original illness;
- (9) the illness was commoner in: (a) hospital staff most closely associated with patients – student nurses 16%, graduate nurses 8%, physicians 6%, attendants 2%, orderlies 2%, (b) females than in males, (c) staff living in hospital residences;

(10) there was no mortality but morbidity was high and 55% of the staff were still off duty 6 months after the peak of the epidemic.

Between 1948 and 1952 Marinacci and Von Hagen (1965) examined twenty-one of these patients (10% of those originally affected), all females, who still had residual muscle pain and fatigue and mental changes. The electromyograms showed generalized, mild, lower motor neurone changes indicative of a radiculopathy, which were quite different from the changes in patients afflicted with paralytic poliomyelitis during the same epidemic. They also found similar changes in a study of over 300 sporadic cases of ENM seen in South California between 1948 and 1965. They observed that recurrences could still occur after 7 years of normal health.

The pathological changes found in patients dying from poliomyelitis in the 1934 Los Angeles epidemic and also in monkeys inoculated with the Californian strain of virus showed differences from those previously reported in poliomyelitis. Destruction of neurones was reduced and there was more marked diffuse perivascular round cell infiltration in the Californian samples.

The conclusions reached by observers as to the aetiology of this epidemic were that the disease was spread by direct personal contact with cases and carriers and not by contamination of the hospital milk or food supply. The illness may have resulted from modification of the disease prevailing in the community, namely, poliomyelitis, or from a coincident infection of a central nervous system infection of unknown aetiology. Hysteria was recognized as a component of the illness, but only in a few cases was it considered to be psychogenic in origin. Other mental disturbances, loss of memory and concentration, and transient personality changes were relatively common.

Subsequent epidemics of ENM displayed the basic features of the Los Angeles outbreak with some variation in clinical presentation and revealed further information about the epidemiology of the disease and its relationship to poliomyelitis.

#### **Military and Hospital epidemics in Switzerland (1937)**

Out of 930 officers and men stationed at Erstfeld, Switzerland, in July 1937, 130 soldiers were affected within 12 days, 108 with the systemic illness and sixteen with meningeal involvement (Table 1). Six soldiers developed an encephalomyelitis with mild muscle paresis. Sweating and hyperaesthetic zones were common. Diminished tone in the limbs was noted in those patients who complained of sharp pain in the legs. The CSF was normal. This benign epidemic did not apparently result in any permanent damage and was recorded on publication (Stahel,

1938) as a 'striking phenomenon for which no analogy could be found in the European literature'. There was no recognized spread to the local civilian population, but another epidemic was recorded elsewhere in Switzerland later in the summer. This involved the separate women's section of the cantonal hospital for St Gall at Frohbürg in

TABLE 1. Frequency of systemic and neurological features of Swiss epidemic, 1937-1939

Type of illness	Place		
	Erstfeld No. of cases (%)	Frohbürg No. (%)	Degersheim No. (%)
Systemic	108 (83)	6 (35)	54 (74)
Meningeal	16 (12)	8 (47)	12 (16)
Encephalomyelitis with paresis	6 (5)	3 (18)	7 (10)

September 1937. Of the twenty-eight patients and staff at risk, seven nurses, five patients, an auxiliary and a male doctor were affected and there were three outside contacts. The age range was from 21 to 38 years and six patients were over 30 years old. Meningeal involvement in eight patients with minimal changes in the CSF and pneumonic changes in five patients were prominent features of the epidemic. Muscle paresis occurred in three patients. The typical prolonged convalescent period was observed with relapses, marked fatigability and autonomic disturbances (Gsell, 1938, 1949, 1958).

One of the interesting features of this epidemic is that the illness was more evenly distributed amongst staff and patients than in other hospital epidemics. Comparison with the military epidemics in Switzerland (Table 1) shows that the involvement of the central nervous system was more than twice as frequent in the hospital cases as in the soldiers. The factor involved may have been the greater opportunity for repeated infection amongst the hospital staff and patients.

#### **Military epidemic at Degersheim in Switzerland (1939)**

In September 1939, 800 officers and men of an infantry battalion arrived at Degersheim from an area in which there was a poliomyelitis epidemic (Gsell, 1949 and 1958). During this month and early October there were seventy-three cases of ENM, fifty-four with the systemic illness, twelve with meningo-neuritic involvement, five with myelitis with muscle paresis and two patients with an encephalitis (Table 1). In most cases the illness was short with low grade fever. The CSF was only slightly abnormal in three of the nineteen cases of meningo-encephalomyelitis. Trigeminal, intercostal and ischial neuralgia were common. Autonomic disturbances were frequently observed in convalescence and the

neurological opinion was that these changes were due to a diencephalitis. Fatigability and loss of concentration persisted in a few cases for more than a year. Some clinicians thought that these symptoms might be a form of neurosis in soldiers anticipating a pension! However, this is the first instance of the use of the term diencephalitis to describe the origin of the autonomic disturbances in the hypothalamus.

This manifestation of ENM was a prominent feature of an epidemic in Lamarque, Texas, U.S.A., in 1965-1966 and was investigated in some depth by Leon-Sótomayor (1969).

There was also a small epidemic among young nurses at Harefield Sanatorium, England, at the same time in 1939 (Houghton and Jones, 1942).

#### District epidemics in Iceland (1948-1949)

On the 25 September 1948 a case of poliomyelitis was reported from the rural area of Akureyri, Iceland, and in the middle of October two further cases occurred, one in the town itself. These patients were severely paralysed and were considered to have classical poliomyelitis. Thereafter, the clinical picture of ENM developed as cases accumulated rapidly, and the disease spread to other districts during December 1948 and January 1949 as indicated in Table 2 (Sigurdsson *et al.*, 1950). In

TABLE 2. Incidence of cases of 'epidemic neuromyasthenia' in Iceland, October 1948 to April 1949

District		Population	Cases	Case incidence
Akureyri	Town	6900	465	6.7%
	Country	2700	23	0.8%
	Total	9600	488	5.1%
Saudárkrókur		2400	147	6.1%
Isafjörður		3020	206	6.8%

Akureyri town the age distribution was predominantly in the 5- to 60-year range with only six cases under 5 years and five cases over 60 years (Table 3). At all ages 5% of the male and 8% of the female population were affected, but there was a high incidence in the 15-19 year group, in which 15% of the males and 18% of the females were taken ill. The major factor in this age group was the large number of cases amongst students at the high school, where 39% of the total cases in the town occurred; 20% of the day pupils and 49% of the resident students of all ages were affected and in the 15-19 age group 60% of the boarders were involved. The systemic form of the illness was present in 70% of patients with the characteristic low fever, muscle tenderness and marked lassitude. The remaining

TABLE 3. Age distribution: percentage of patients in each age group in three District outbreaks

Age group (years)	District		
	Akureyri	Dalston	Adelaide
0-4	1	12	15
5-9	4	25*†	20
10-19	37†	12*	20
20-29	21	8	20
30-49	30	21	25
50-65	7	22	

\* Dalston age groups were 5-11 and 12-19 years.

† Percentages in *italic* indicate the presence of an institutional outbreak in the age group.

30% had muscle weakness with pyrexia frequently over 38°C (100.4°F). Ulnar and shoulder girdle neuritis was described as well as radiculitis involving the T1, L5 and S1 nerve roots. Mental changes including hysterical episodes were observed. Subacute arthritis was noted in the convalescent period, during which multiple relapses occurred in some patients with renewal of fever and muscle tenderness and weakness at a new site. The CSF was examined in eight patients and showed slightly raised cell and protein readings in 50% of the cases. Virus studies failed to involve poliomyelitis, Coxsackie or known encephalitis viruses in the epidemic.

In 1955 Kjartan Gudmundsson, a neurologist, re-examined thirty-nine patients affected by the 1948 epidemic in Akureyri (Sigurdsson and Gudmundsson, 1956) and found that of *those more severely affected* only 25% had completely recovered, 52% had residual muscle tenderness, and 65% had objective neurological signs. Many patients still complained of nervousness, abnormal fatigability of muscles, muscle pain, sleeplessness and loss of memory. *Of those mildly affected* in 1948 only 44% had fully recovered, 50% had muscular tenderness, and 19% had residual objective neurological signs.

In the same year there was an epidemic of type-1 poliomyelitis in Iceland, which spread around the coast (see Fig. 1). It failed to become established at Saudárkrókur and Akureyri and changed to show features of ENM at Patreksfjörður (135 cases of ENM) and Þórshöfn (114 cases). The clinical pattern was confirmed by the finding of antibody to type-1 poliomyelitis in 50-95% of the school-children in areas affected by poliomyelitis and no antibody in children in areas affected by ENM. However, children in ENM areas responded to poliomyelitis vaccination with higher antibody titres than in other areas not affected by the poliomyelitis epidemic, as if these children had already been exposed to an agent immunologically similar to poliomyelitis virus (Sigurdsson, Gudnadóttir and

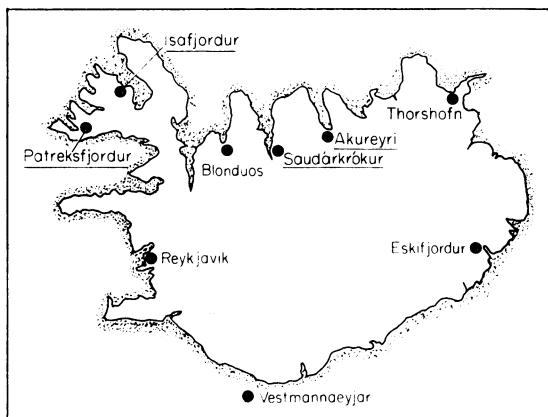


FIG. 1. Iceland, showing the towns in the south affected by the epidemic of type-1 poliomyelitis in 1955 and towns on the north coast (underlined) in which outbreaks of 'epidemic neuromyasthenia' occurred in 1948 and 1955.

Pétursson, 1958). Thus, the agent responsible for ENM would appear to be able to inhibit the pathological effects of poliomyelitis infection. When an American airman was affected in the 1955 epidemic and returned home, a similar secondary epidemic occurred in Pittsfield, Massachusetts, U.S.A. (Hart, 1969; Henderson and Shelokov, 1959).

#### District epidemic in Adelaide, Australia (1949–1951)

An epidemic of poliomyelitis commenced in May 1949 in Adelaide, Australia. In August, cases of ENM began to appear and continued to be seen until 1951, by which time 800 patients had been admitted to Northfield Infectious Diseases Hospital (Pellew, 1951). The age incidence is shown in Table 3. The male : female ratio was 1 : 1. In contrast to the poliomyelitis cases being admitted at the same time, the CSF examination revealed normal cellular content and protein in the majority of cases of ENM. The record of the monthly totals showed that the number of acellular ENM cases remained high during the early months of 1950 as the number of classical cellular poliomyelitis cases waned (Jackson, 1952). During the second summer of 1950–1951 the monthly incidence suggests that once the epidemic of ENM had been triggered off, its natural seasonal virulence closely paralleled that of poliomyelitis. Once again cases of pneumonitis occurred. The muscle weakness was described as slight, diffuse and mainly affecting the legs, especially the tibialis anterior and gluteus medius muscles. In some patients the muscle weakness appeared for the first time up to three months after the onset of the initial symptoms. Patients were particularly liable to relapse during the hot weather the following summer.

Although investigations for the viruses known at that time were negative, an agent was repeatedly transmitted to monkeys from two patients (Pellew and Miles, 1955). When the monkeys were killed minute red spots were observed along the course of the sciatic nerves. Microscopically infiltration of nerve roots with lymphocytes and mononuclear cells was seen and some of the nerve fibres showed patchy damage to the myelin sheaths and axon swellings. Similar findings had been produced by the transmission of an agent to monkeys from a child with poliomyelitis in Boston, Massachusetts, in 1947 (Pappenheimer, Cheever and Daniels, 1951). However, in these monkeys the changes were more widespread, involving the dorsal root ganglia, cervical and lumbar nerve roots and peripheral nerves. Perivascular collars of lymphocytes and plasma cells were seen in the cerebral cortex, brain stem and cerebellum, spinal cord and around blood vessels to the nerve roots. There was no evidence of damage to the nerve cells in the brain or spinal cord. The distribution and intensity of the lesions varied considerably from monkey to monkey. This pathological picture of mild diffuse changes corresponds closely to what might be expected from clinical observations of patients with neurological involvement in ENM.

#### District epidemic in New York State (1950)

In the autumn of 1950 there was a widespread epidemic of poliomyelitis in New York State (U.S.A.). At the same time White and Burtch (1954) discovered thirty-three patients with ENM, which they called Iceland disease and studied seventeen patients in detail. The age distribution was from 9 to 45 years. Lymphadenopathy was recorded for the first time in five patients. Ulnar and sciatic nerve lesions were reported. Twelve out of thirteen patients tested showed a raised urinary creatine excretion. Raised urine creatine excretion and creatine : creatinine ratios (up to 145% – normal up to 6%) were noted in four patients during an outbreak of ENM in a convent in New York State in 1961 (Albrecht, Oliver and Poskanzer, 1964). [The author has recently observed raised creatine : creatinine ratios during a major relapse of ENM in a patient whose illness has taken an episodic course since the initial illness in 1961. Her serum creatine phosphokinase was normal.]

Subsequent epidemics have occurred as recorded in Table 4 (Henderson and Shelokov, 1959). The Danish cases include the ten patients described by Fog (1953) and other patients mentioned by Pederson (1956) in a survey of encephalitis in Jutland and classified as encephalitis with vertigo. The clinical picture of ENM can be clearly recognized. Abnormal lymphocytes were observed in peripheral

TABLE 4. Outbreaks of 'epidemic neuromyasthenia' 1950-55

Year	Place	Number of cases	Nature of epidemic
1950	Louisville, Kentucky, U.S.A.	37	Student nurses
1952-54	Denmark	over 70	District
1952	Lakeland, Florida, U.S.A.	over 27	District
1952	Middlesex Hospital, London	14	Student nurses
1953	Coventry, England	over 13	Hospital staff
1953	Rockville, Maryland, U.S.A.	50	Student nurses
1954	Tallahassee, Florida, U.S.A.	450	District
1954	Seward, Alaska	175	District
1954-55	Johannesburg, South Africa*	14	District
1955	Durban, South Africa	140	Hospital staff
1955	Berlin, Germany	7	Military
1955	Boscombe, England†	2	Hospital staff

\* Jackson, Jakobson and Cooper (1957).

† Jelinek (1956).

blood and sternal marrow studies. In the Durban outbreak a number of urines were tested for toxic substances and some contained a non-nitrogenous acidic metabolite, which was toxic for the larvae of *Aedes aegypti*. Apparently the substance is present in small amounts in normal urines, but it could not be identified (Hill, Cheetham and Wallace, 1959). No subsequent investigation of this abnormality has been reported. The epidemic at Rockville, U.S.A. (Shelokov *et al.*, 1957), was associated with the isolation, from a number of the ENM patients, of Bethesda-Ballerup paracolon organisms which appear to have triggered off the disease in the same manners as infection with poliomyelitis virus precipitated other epidemics.

There was an extensive outbreak of ENM in the North of England in 1955. The first case was recorded in a male adult by the principal of a general practice covering the villages of Dalston, Orton and Thursby, Cumbria, on the 16th January (Wallis, 1957). The villages are situated between Carlisle and the foothills of the Lake District. By the beginning of February there was an extensive epidemic in the primary school children and the spread to the adults took place in March and April. The epidemic continued until July, by which time 233 cases had been recorded out of the practice population of 1675 children and adults, a case incidence of 14%. The age distribution is shown in Table 3. The male : female ratio was 1 : 1. The peak incidence of 33% was recorded in males in the 5-11 year age group, and the highest female incidence (26%) occurred in the under 5 year age group. Twenty children boarded at the Carlisle Corporation Home, Dalston, mainly boys aged 5 to 15 years, were all affected. Secondary attacks in contacts occurred in several families after a primary host relapsed, suggesting that the patient had again become infectious. Tenderness on palpation of enlarged lymph glands and over the liver and

spleen was common, especially in children, but jaundice was rare. In approximately 30% of the blood films there were morphological changes in the lymphocytes with relative lymphocytosis and eosinophilia. The blood picture was different from that seen in glandular fever and the Paul Bunnell tests were negative. In some cases the abnormal blood films persisted for at least 18 months. [Recently abnormal lymphocytes were found in one of the author's own patients during a relapse 16 years after the onset of her illness.] In most of the Dalston cases the onset was abrupt after an incubation period of 5 to 7 days, but some of the adults showed a delayed onset with the illness reaching the maximum severity after 6 weeks. Neurological symptoms occurred in 60% of the patients, but objective changes were mild and seen only in 20% of cases. There were ulnar nerve lesions in eight patients. Non-specific abnormalities were noted in the EEGs in fifteen of the twenty-three patients tested. The CSF was normal in the few examinations made. Virus studies were also negative. The case incidence and percentage of neurological involvement were similar to that observed in the Swiss military epidemics.

By July 1955 the disease had spread eastwards to County Durham, where many additional lower motor neurone lesions, particularly ulnar nerve palsies, were seen in the electrodiagnostic clinic. The general physicians in the area mentioned that they were dealing with a form of viral hepatitis in the community, in which jaundice was rare, but in which anxiety, depression and other mental changes occurred during convalescence. The abnormal blood films were also reported during a similar outbreak affecting a boys' school at Sedburgh, Yorkshire.

The characteristic features of the Dalston epidemic can be summarized as follows:

(1) a systemic illness with relatively low fever or subnormal temperatures in delayed onset cases and tenderness over enlarged lymph glands, liver and/or spleen;

(2) marked fatigability;

(3) mental changes, such as impairment of memory and concentration, changes of mood with behaviour disorders in children, sleep disorders, irritability or depression;

(4) involvement of the autonomic nervous system resulting in orthostatic tachycardia, coldness of the extremities, episodes of sweating or profound pallor, sluggish pupillary responses, constipation and frequency of micturition, possibly as the result of a hypothalamic disturbance;

(5) diffuse and variable involvement of the nervous system leading to ataxia, weakness or sensory changes in a limb, nerve root or a peripheral nerve distribution, especially involving the ulnar nerve;

(6) muscle pain, tenderness and myasthenia;

(7) recurrences in about 20% of patients over a period of several years.

### Conclusions

This review of the early outbreaks of ENM reveals the wide spectrum of the presentation of the illness from a mild systemic disturbance, which is similar to that of other enteroviral diseases, to a meningo-encephalomyelitis with ocular palsies and upper motor neurone signs. However, the systemic illness is distinguished in many patients by a prolonged convalescence portrayed by mental changes, particularly depression, autonomic disturbances, a profound tendency to fatigue easily and relapses of the original features of the illness. The neurological complications occur more frequently in confined outbreaks affecting large groups of persons living together. The neurological changes vary considerably in situation and severity from patient to patient and show a similar tendency to recur at the original site or in a different part of the nervous system. Several epidemics appear to have been triggered off by an outbreak of an infection with enteric organisms or poliovirus which then subsides. In some patients there is a direct involvement of muscles, which may feel swollen and tender and fatigue rapidly on sustained activities such as walking. The profound generalized and muscular fatigability is a striking feature of the disease. Whether the toxic urinary metabolite discovered in the Durban outbreak is connected with this phenomenon has yet to be determined.

A strange feature of this disease is the frequent occurrence among young female nurses in hospital epidemics, while patients are usually unaffected. The factors involved in this situation are (1) the epicentre of the outbreak is in the nurses' residence

rather than in the hospital wards, (2) the nurses may become infectious during recurrences and the whole residential community may be subjected to multiple exposure to infection, and (3) the deleterious effect of physical exercise during exposure and convalescence. The unusually high incidence of patients affected in the women's ward of the Frohburg (St Gall) hospital may have been due to the probability that some of the women were in labour when exposed to infection.

Neither of the names commonly used for the disease is completely satisfactory. 'Epidemic neuromyasthenia' describes the epidemic nature of the disease and the abnormal fatigability of the nervous system and muscles, but the term is liable to be confused with neurasthenia and does not take into account the encephalomyelopathy. 'Benign myalgic encephalomyelitis' does not define the mild systemic form of the disease, which occurs frequently in district epidemics with its characteristic fatigability. The chronic disability revealed by the follow-up of the Los Angeles cases does not justify the title 'benign'. However, until more is known about the underlying pathology of the disease it seems preferable to use the term 'epidemic neuromyasthenia' and qualify the more severe neurological cases with the description 'encephalomyelitic form'.

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